Beyond the Headlines: Why Henipaviruses Warrant Our Attention

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ABSTRACT

Henipaviruses, including Hendra and Nipah viruses, represent significant zoonotic threats with higher mortality rates. Due to limited therapeutic interventions, poses substantial challenges. These bat-borne pathogens were first identified in Australia (Hendra, 1994) and Malaysia (Nipah, 1998–1999), with subsequent multiple outbreaks. The recent discovery of Camp Hill virus in North American shrews, suggest broader geographic distribution than previously recognized.

Transmission occurs primarily through contact with reservoir hosts, though human-to-human spread has been documented in Nipah outbreaks. Initial non-specific febrile symptoms can progress to fatal encephalitis with distinctive pathological findings including syncytia formation and vasculitis. A concerning feature is the potential for relapsing encephalitis months or years after initial infection.

Management remains predominantly supportive, highlighting the urgent need for effective antivirals, vaccines, and enhanced surveillance. Expanded research into therapeutic countermeasures is essential to address this emerging global public health threat.

KEYWORDS

henipavirus; camp hill virus; Pteropus; hendra virus; nipah virus; encephalitis; flying fox; outbreak; shrew

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INTRODUCTION

Highly pathogenic henipaviruses, which are characterized by their zoonotic origin, has been an evolving threat to human health and raise concern due to it limited therapeutic options. Hendra virus (HeV) and Nipah virus (NiV), members of genus henipaviruses, has high mortality rates around 60% and 92% respectively in humans (1). These HeV and NiV are bat-borne viruses whose clinical presentation may range from mild influenza-like symptoms to complications such as severe encephalitis and/or respiratory failure (2).

HISTORY

In 1994, the first reported case of henipavirus was reported in Australia when HeV caused a severe respiratory disease eruption in horses, concomitantly resulting in a few human fatalities (2). Soon after, in 1998–1999, an epizootic outbreak emerged following the identification of NiV in 283 human cases in Malaysia, which caused severe encephalitis with high mortality in 109 humans with and notable respiratory problems in pigs which served as amplifying intermediate host (2, 3). Since these initial outbreaks, there has been the progressive emergence of henipaviruses across wider geographic areas, particularly in the Asia-Pacific and African continents with significant public health consequences.

Smaller cluster outbreaks, although with higher mortality rates of NiV have primarily occurred in Bangladesh and India since 2001. Several other novel henipaviruses have been subsequently identified, including Cedar virus (CedPV), which is also a bat-borne virus that is non-pathogenic to animals and has been studied to be non-zoonotic (1, 2). Mojiang virus (MojV) was initially detected in a Rhinolophus cave-dwelling rats in China following three miners' death in 2012 from a severe pneumonia with acute respiratory distress syndrome. Furthermore, during the period from 2018–2022, multiple febrile patients in China were documented to have a Langya virus (LayV), a phylogenetically distinct shrewborne henipavirus that demonstrates significant zoonotic potential (4).

In January 2025, research team from the University of Queensland and Auburn University reported the first detection of a henipavirus, specified as Camp Hill virus (CHV) in North America. This virus was identified through the analysis of tissue samples obtained from four dead northern short-tailed shrews in 2021 from Tallapoosa County, Alabama (5). While no human infections with CHV have been documented thus far in epidemiological investigations, phylogenetic analysis revealed that CHV is genetically related to LayV which has previously demonstrated capacity for cross-species transmission from shrews to humans in China (5).

GENOMIC

Henipaviruses belongs to the Paramyxoviridae family (1). On a molecular level, henipaviruses exhibit a complex molecular structure and replication process that contribute to its significant pathogenicity. The viral genome is a single-stranded, negative-sense RNA approximately 18.2 kb in length which encodes six essential structural proteins (1). This genomic architecture supports the complexity of the viral life cycle and host interaction mechanisms. Henipavirus entry relies on a dual-protein system, in which the glycoprotein (G) binds to ephrin B2/3 receptors on host cells. These receptors are highly maintained across mammals and widely expressed in vascular endothelium and neurons (6, 7). Upon G protein binding, conformational changes activate the fusion (F) glycoprotein, which exists in a metastable pre-fusion state following proteolytic cleavage during virion maturation (8). In its activated form, the F protein undergoes structural reorganizations, exposing its hydrophobic fusion peptide. The insertion of this peptide into the host cell membrane initiates the formation of a fusion pore via which the viral ribonucleocapsid enters the host cell cytoplasm.

The virion's core comprises a ribonucleocapsid complex, in which nucleocapsid (N) proteins encapsulate the viral RNA genome that helps in protection and serve as a template for viral RNA synthesis (1). The ribonucleocapsid associates with the phosphoprotein (P) and large polymerase protein (L) to constitute the functional replication complex. During the viral budding process, a matrix (M) protein layer surrounding the viral ribonucleocapsid facilitates the interaction between the viral core and the host-derived envelope (7).

The viral P gene also encodes three non-structural proteins (C, V, and W) that are expressed in infected cells. These accessory proteins play a role in immune evasion mechanism, with the V protein specifically binding signal transducers and activators of transcription (STAT) molecules to prevent their nuclear translocation without degradation (7, 8). The W and C proteins contribute to immune evasion through mechanisms that are not yet fully understood.

TRANSMISSION

The spread of henipavirus between species occurs through multiple routes within the ecological setting. Fruit bats of the genus Pteropus (flying foxes) are known as the primary natural reservoir hosts for this virus, typically harboring viruses without exhibiting signs of disease (9). Although the infectivity of henipavirus is lower compared to that of the recent coronavirus (COVID-19) pandemic, its mortality rate is much higher-exceeding 60% for henipavirus, compared to mortality rate of less than 1% for COVID-19 globally (1, 10).

Several mechanisms have been explained for henipavirus transmission among species (Fig. 1).

SPILLOVER TRANSMISSION

There are multiple pathways that facilitate this spillover of henipavirus from the reservoir hosts. A Pteropid bat has been documented in most cases with direct and indirect spillover events (9). Bat behaviors such as roosting and

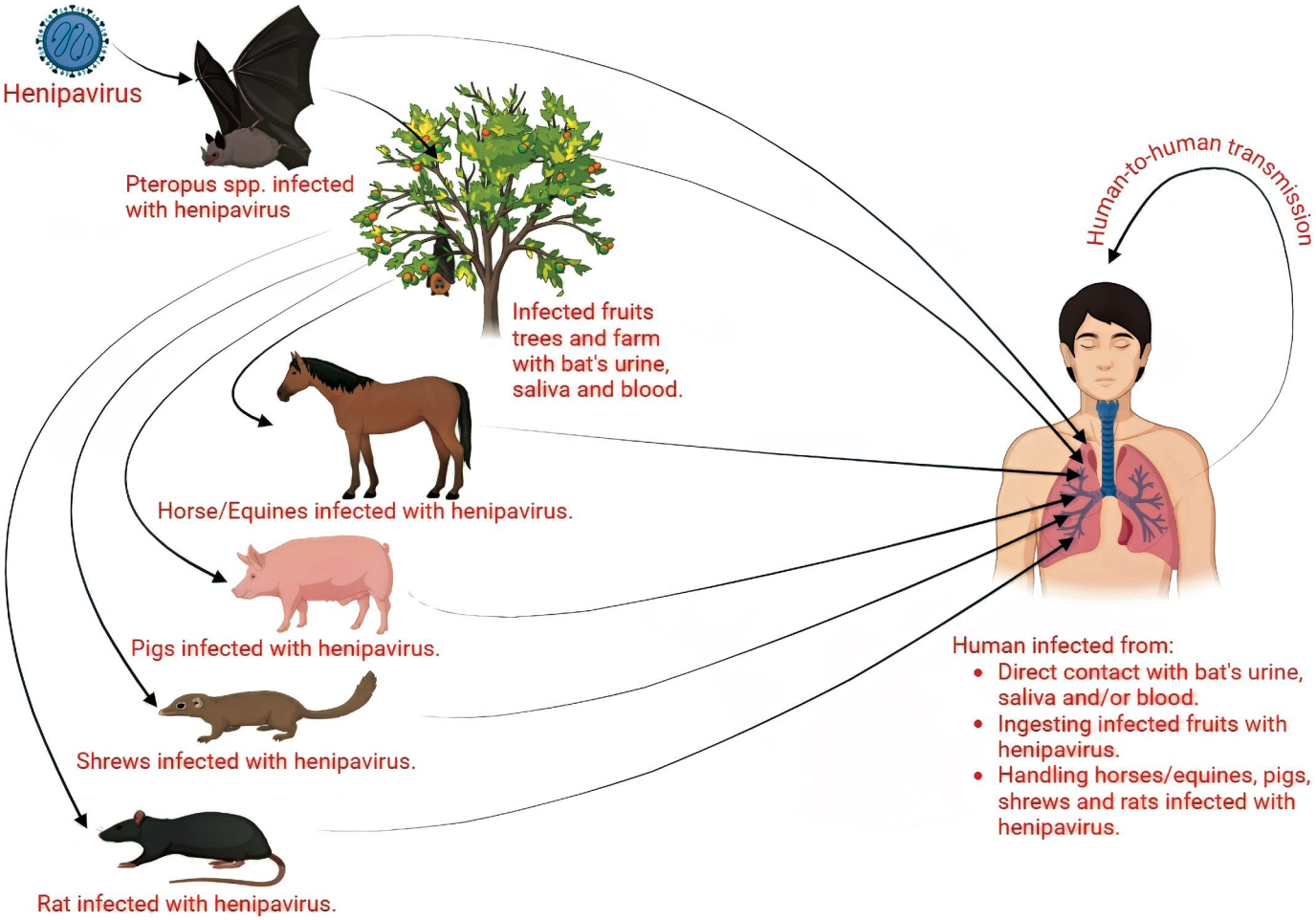

Fig. 1 Illustration of henipavirus transmission from its natural host, fruit bats (*Pteropus* spp.), to susceptible species. The arrows represent virus transmission within the natural reservoir and show up spillover events leading to disease.

feeding patterns create opportunities for virus shedding into the surrounding environment through saliva, urine, and/or excreta. These infectious materials may contaminate food sources consumed by intermediate hosts or humans, as it was reported during the transmission of NiV through palm sap consumption in Bangladesh (11).

Among domesticated animals, particularly pigs and horses have played significant roles as amplifying intermediate hosts in historical henipavirus outbreaks (1). In the case of NiV in Malaysia, intensive pig farming practices led to rapid viral transmission among swine populations, subsequently leading to human infections among farm workers and others in contact with infected animals (1). Likewise, HeV outbreaks in Australia was associated with horse infections followed by human transmission those in close contact with ill equines (1). These intermediate hosts often exhibit enhanced virus shedding and may facilitate viral adaptation through selective pressures, potentially increasing transmissibility to humans.

The molecular basis for interspecies transmission capability lays partly in the conservation of ephrin B2/B3 receptors across mammalian species (8). Additional factors such as viral genetic adaptations, host immune status, and ecological conditions collectively determine the success of cross-species infection. Relatively few mutations are reported to be required to enhance henipavirus transmission in new host species, emphasizing the evolutionary plasticity of these viruses and their potential for adaptation to different cellular environments (8, 12).

HUMAN-TO-HUMAN TRANSMISSION

Secondary route of transmission thru human-to-human has been documented in several NiV outbreaks in Bangladesh and India (11). This characteristic for sustained human transmission represents a concerning aspect of henipavirus epidemiology, as it eliminates the requirement for continued animal exposure once the virus has entered human communities. Close contact with infected individuals, particularly through caregiving activities, presents the highest risk for secondary transmission (1, 11).

Viral shedding through respiratory secretions, saliva, and other bodily fluids has been reported for human-to-human transmission. Patients with respiratory involvement may generate infectious aerosols during coughing or sneezing, facilitating airborne transmission in close-contact settings. Additionally, direct contact with infectious bodily fluids from patients with encephalitis or other symptoms can lead to transmission through mucous membrane exposure or percutaneous inoculation (1). Healthcare settings have emerged as significant amplification points for human-to-human spread, with nosocomial transmission documented in multiple outbreaks. Particularly when managing patients with suspected henipavirus

infections, it is important to follow proper infection control practices (11).

The reproductive number (R0) for human-to-human transmission of NiV has been estimated at approximately 0.5 in community settings, suggesting that sustained chains of transmission are unlikely under normal circumstances (11, 13). However, super spreading events, wherein a single infected individual transmits to an unusually large number of secondary cases, have been observed. Such events are influenced by factors including viral load, symptomatic presentation, and culturally specific practices around illness and death. The potential for viral adaptation to enhance human-to-human transmissibility remains a significant concern, as relatively minor genetic changes could potentially increase the R0 above the epidemic threshold of 1.0, leading to sustained transmission chains and larger outbreaks (11, 13).

ECOLOGICAL TRANSMISSION

Natural habitat destruction and land-use changes have disrupted bat foraging patterns; this has increased the frequency of bat-human interfaces. Specifically, increasing number of Pteropus bats has been seen to utilize agricultural area and human settlements for feeding and roosting (11). The migratory pattern of the bat due to climate change further compounds these effects, expanding their geographical range (Fig. 2), and potentially stressing bat populations in ways that may enhance viral shedding (14).

The establishment of large-scale pig farms and agricultural land in regions overlapping with flying fox habitats has created conditions conducive to viral amplification and human exposure (11). The Malaysian Nipah outbreak of 1998–1999 exemplifies this dynamic, wherein pig farms established beneath fruit trees frequented by bats created an ideal setting for virus introduction, amplification, and subsequent human infection (11). The practice of date palm sap collection in Bangladesh similarly represents an anthropogenic activity that creates a transmission pathway, as bats visiting the sap collection pots contaminate the sap with virus-containing saliva and urine, which is then consumed by humans without processing that would inactivate the virus (11).

Socioeconomic factors, like limited healthcare infrastructure, inadequate surveillance systems, and cultural practices surrounding caregiving and burial rituals can further facilitate viral spread once human infections occur (1, 11). Additionally, ecological encroachments, such as deforestation for agricultural expansion, create feedback loops that intensify spillover risk while constraining the resources available for prevention and response measures.

CLINICAL PROGRESSION

A variable course has been suggested for clinical progression, ranging from asymptomatic infection to severe fatal encephalitis, with distinctive pathophysiological mechanisms involving vascular damage, immune evasion, and potential for latent infection with delayed recrudescence.

INCUBATION PERIOD

Henipavirus infections typically manifest after an incubation period ranging from 4–14 days in humans, though in some rare cases it has been reported to extend up to 45 days (16). This variability in incubation period depends on multiple factors including viral dose, route of exposure, and host factors (17).

INITIAL PRESENTATION

Clinical manifestations resemble influenza-like illness initially, characterized by abrupt onset of fever, headache,

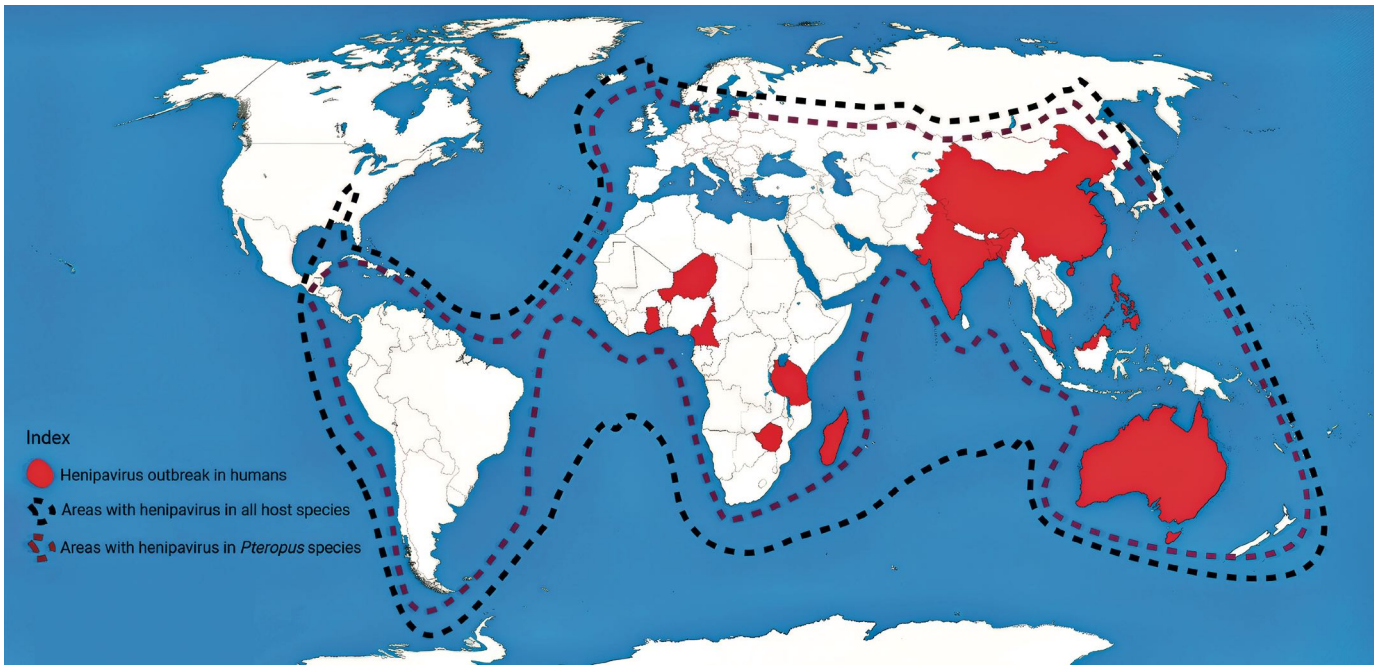

Fig. 2 Geographic distribution of regions inhabited by host species carrying henipavirus and human henipavirus outbreaks.

dizziness, and vomiting, typically followed by myalgia and general malaise (15). These non-specific prodromal symptoms make early diagnosis challenging, particularly in regions where other febrile illnesses such as malaria or dengue are endemic. The initial viral replication occurs in the respiratory epithelium before the virus disseminates systematically through endothelial cells entering the bloodstream (1).

Early phase respiratory symptoms include mild cough and sore throat, which can rapidly progress to more severe respiratory distress in some cases. The virus is known to be shed in nasal secretions even before the onset of symptoms, with evidence showing that horses can shed HeV in nasal secretions as early as two days post-exposure, prior to developing clinical signs (17). This pre-symptomatic shedding contributes to the transmission dynamics of these viruses and presents significant challenges for infection control during outbreaks. Laboratory findings typically shows non-specific changes such as leukopenia, thrombocytopenia, and elevated liver enzymes during the early clinical presentation, which lacks disease-specific pathognomonic features.

MECHANISMS OF TISSUE DAMAGE

The pathogenesis of henipavirus infection is linked to the virus's cellular tropism, which is determined by the distribution of its entry receptors, primarily ephrin-B2 and ephrin-B3 (8). These receptors are predominantly expressed in many tissues including endothelial cells, neurons, and respiratory epithelium; this explains the systemic nature of infection and the broad host range of henipaviruses compared to most other paramyxoviruses (19). This widespread receptor distribution facilitates viral dissemination to multiple organs and tissues throughout the body. Initially, direct viral damage to endothelial cells lining blood vessels, leads to a characteristic vasculitis observed in multiple organ systems.

A distinctive pathological feature of henipavirus infection is the formation of multinucleated giant cells known as syncytia (1). This occurs when viral glycoproteins expressed on the surface of infected cells bind to cellular receptors on neighboring cells, triggering membrane fusion mediated by the viral F protein (1). The resulting syncytia formation is associated with extensive tissue damage, including necrosis, vasculitis, and thrombosis in affected organs. Autopsy findings from NiV-infected patients have revealed widespread vasculitis in the lungs (62%), kidney (24%), heart (31%), and central nervous system (80%), correlating with the expression pattern of ephrin-B2 in these tissues (20). Additionally, necrosis is commonly observed in highly vascularized organs such as the spleen, particularly in regions expressing ephrin-B2 (1, 19). These pathological changes explain the clinical manifestations of respiratory disorders, neurological symptoms, and hemodynamic instability which are observed in henipavirus infections.

NEUROLOGICAL MANIFESTATIONS

Neurological manifestations become increasingly prominent when henipavirus infection progresses and often define the severe stage of disease. The virus can enter the central nervous system (CNS) through multiple routes: via infected endothelial cells of the blood-brain barrier, through direct infection of olfactory neurons from the nasal cavity, or via retrograde axonal transport (1, 21). Viral replication in the CNS leads to neuronal damage, inflammation, and disruption of the blood-brain barrier, resulting in progressive neurological impairment. Particularly in patients with reduced levels of consciousness, clinical neurological manifestations exhibit altered consciousness, areflexia, hypotonia, and/or abnormal doll's eye reflex (15, 20).

Neurological signs indicating acute encephalitis have been reported in more than 70% of cases, with severe weakness in 67% and areflexia or hyporeflexia in 65% (20). A distinctive and diagnostically significant finding seen in approximately 30% of NiV encephalitis patients is segmental myoclonus, which involves diaphragm and muscles in the limbs, neck, and face (20). Other neurological manifestations include meningism, generalized tonic-clonic seizures, nystagmus, and cerebellar signs, indicating the widespread involvement of different parts of the nervous system (22). In severe cases, progressive neurological deterioration leads to coma and death, with overall mortality rates ranging from 40% to 70% (21, 22).

RELAPSING AND LATE-ONSET ENCEPHALITIS

Perhaps one of the fascinating and clinically significant aspects of henipavirus infections is the occurrence of relapsing or late-onset encephalitis, which can develop weeks, months, or even years after the initial infection (21, 23). This phenomenon has been documented in both HeV and NiV infections, though it appears to be more common with NiV. Relapsing encephalitis may affect up to 10% of survivors and can occur following either symptomatic infections (ranging from mild illness to acute encephalitis) or even after asymptomatic seroconversion (15, 21). The most delayed case documented occurred 11 years after an asymptomatic infection, underscoring the potential for long-term viral persistence in the CNS (21).

Clinical and radiological observations indicate that encephalitis caused by recurring henipavirus infection presents differently from acute encephalitis (21). Magnetic resonance imaging in the relapsing phase typically reveals more extensive and confluent hyperintense cortical lesions compared to those documented during the acute phase. Pathologically, relapsing encephalitis is distinguished by widespread and confluent necrosis of the parenchyma, edema, and inflammation, primarily affecting neuronal regions. This condition is also characterized by prominent perivascular cuffing, severe loss of neurons, reactive gliosis, and neovascularization (15, 21). Viral inclusions, antigens, and RNA are predominantly detected in surviving neurons. In the relapsing form, the vasculitis, endothelial syncytia, and thrombosis typical of acute henipavirus encephalitis are absent. Furthermore, blood vessels do not contain viral antigens or RNA, suggesting that relapsing encephalitis results from the reactivation of latent viral foci within the central nervous system rather than reinfection or entry from outside the

CNS (21). The estimated case fatality rate for relapsed and late-onset Nipah virus encephalitis is approximately 18%, which is significantly lower than that of acute encephalitis (23).

DIAGNOSTIC

The diagnosis requires a combined clinical evaluation with laboratory confirmation. Clinical assessment should focus on identifying initial characteristic symptoms including encephalitis, fever, headache, respiratory distress, seizures, and altered mental status, particularly in patients with relevant exposure history in endemic regions (24). First-line diagnostic investigations include serology, RT-PCR, complete blood count, liver function tests, serum electrolytes, clotting profiles, chest radiography, and cerebrospinal fluid analysis (24, 25). For challenging cases, advanced imaging (MRI), electroencephalography, and rare brain biopsy may be necessary (24).

In the laboratory setting, diagnosis is made through several methods, with nucleic acid amplification testing (NAAT) serving as the gold-standard for definitive diagnosis due to its high sensitivity, detecting as few as 20 viral genomes (25). Reverse transcription polymerase chain reaction (RT-PCR) targets the conserved viral N, M, or P genome segments, can rapidly identify viral RNA in clinical specimens including throat and nasal swabs, cerebrospinal fluid, blood, urine, and respiratory secretions (25). RT-PCR demonstrates excellent sensitivity, detecting between 500–1000 copies of RNA templates with the lowest detection threshold at 0.37 pg/μL of RNA (24).

Enzyme-linked immunosorbent assay (ELISA) can detect both viral antigens and host antibody responses (IgM and IgG), typically requiring 3–4 hours for completion and applicable to both human and animal samples (24, 25). IgM antibody detection has been reported to peak approximately nine days after illness onset and can persist for at least three months, while IgG peaks after 17 days and remains detectable for more than eight months (25). Especially, in resource-limited settings where PCR facilities may be unavailable, serological testing complements molecular can be diagnostics. Recognition of the diagnostic challenges in rural and remote settings has prompted the development of point-of-care and “near-POC” NAAT platforms requiring minimal infrastructure and training, potentially tackling outbreak management capabilities in resource-limited areas (24).

MANAGEMENT

Henipavirus infections management remains predominantly supportive due to the absence of approved specific antiviral therapies, this presents significant challenges given the high mortality rates associated with these infections (24, 26). The primary treatment involves supportive care, focusing on fundamental clinical procedures: maintaining fluid and electrolyte balance, prophylaxis against venous thrombosis, ensuring airway patency, and mechanical ventilation for respiratory compromise (24). Broad-spectrum antibiotics are typically administered to prevent secondary bacterial infections that might complicate the clinical course (24). Ribavirin has been documented to yield promising results, reducing mortality by approximately 36%, when used empirically during the 1998–1999 Malaysian outbreaks (26). However, evidence remains limited due to the open-label nature of the studies and their reliance on historical controls. The broad-spectrum antiviral remdesivir demonstrated protection in African green monkeys when administered within 24 hours of NiV exposure and continued for 12 days (26).

Several experimental therapeutics shows encouraging potential for future treatment protocols. Favipiravir (T705) exhibited protective effects in Syrian golden hamsters, while chloroquine demonstrated efficacy in suppressing viral replication in cell cultures, though it’s in vivo efficacy remains uncertain (24, 26). Particularly promising are biologics such as the monoclonal antibody m102.4, which has demonstrated protection in animal models and has progressed to Phase I human trials with manageable adverse events, yielding encouraging results (24). Passive immunotherapy approaches using monoclonal antibodies have protected ferrets against NiV infection and hamsters from HeV (27). Novel compounds such as Griffithsin (GRFT) and its synthetic derivatives have demonstrated significant inhibition of viral replication, with in vivo evaluations in golden Syrian hamsters showing protection against lethal NiV challenge (24). Although progress has been made, the treatment window remains narrow, usually within 24 hours of exposure, emphasizing the critical need for rapid diagnostics and accessible treatments to improve survival rates (26). For patients who recover, long-term follow-up remains essential due to the potential for relapsing encephalitis and neurological sequelae.

CONCLUSION

The recent discovery of CHV in North America represents a significant expansion in our understanding of henipavirus distribution globally. This finding demonstrates that these potentially deadly pathogens are not confined to their previously known ranges in Asia, Australia, and Africa, but may be more widely distributed throughout the world.

The high fatality rates associated with henipaviruses like Nipah and Hendra emphasizes the importance of proactive research and preparedness efforts. Public health systems should remain vigilant for unusual disease patterns, particularly in regions where known intermittent hosts are common.

As humans continue to encroach on wildlife habitats and climate change alters ecological relationships, the emergence of novel pathogens becomes increasingly likely. The CHV discovery serves as a timely reminder of the ongoing need for robust disease surveillance systems and coordinated international responses to emerging infectious disease threats before they develop into epidemic or possible pandemic.

AUTHORS' CONTRIBUTIONS

Biplab Adhikari: Conceptualization, data acquisition, formal analysis, supervision, drafting the original manuscript, creating diagrams and figures, reviewing, and editing and submission of final version.

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